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Case Report

Acute Common Femoral Artery Occlusion following Total Hip Arthroplasty for an Arthrodesed Hip — A Case Report —

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Abstract: Perioperative acute arterial complications occur rarely after total hip arthroplasty (THA) but can be limb or life threatening. Here, we report a case of an arterial thrombosis after primary THA for an arthrodesed hip. The arterial occlusion occurred because of the surgical mobilization against the immovable flexion and adduction position. A review of the literature on vascular complications arising after THA suggests multiple possible mechanisms and clinical presentations that relate to these complications. Specific risk factors can be identified in THAs replacement cases and THA for the arthrodesed hip can be a major risk factor. Most of these vascular complications can be prevented or more efficiently treated by thorough preoperative assessment and careful postoperative monitoring.

Key words: arthrodesis, thrombosis, femoral artery injury, THA

Introduction

Vascular complications related to total hip arthroplasty (THA) are rare[1)] and the majority are secondary to direct injury by the retractors, removal of extruded cement during revision surgery, and acute or chronic migration of prosthetic components[2-8)]. Similarly, arterial thrombosis is rarely reported after THA. The external iliac artery is commonly involved and appears to be associated with lengthening of the limb and leg twisting that occurs during the posterior approach, or correction of a severe hip flexion contracture[9-10)]. Occlusion of the superficial femoral artery or the iliofemoral artery has been reported less commonly. Here, we report a case of common femoral artery (CFA) thrombosis during THA for an arthrodesed hip.

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Case Report

A 67-year-old female consulted us requesting the mobilization of her arthrodesed left hip. Her past history included hypertension, hyperlipidemia, and acute myocardial infarction. She also had a history of infectious arthritis for her left hip at the age of two and had received an arthrodesis at the age of ten. Subsequently she had few problems with her hip except for a severe limp. At 64 years of age she was admitted to hospital for acute myocardial infarction and had successful interventional treatment. The long-term hospitalization associated with this treatment caused a reduction in her physical strength, and decreased her activities of daily life. After that time she needed double canes, became unable to walk more than 10 meters and had difficulty sitting down in a chair. At our consultation, her left hip was arthrodesed in an almost functional position ; 35° of flexion, neutral in rotation, and 10° of adduction (Fig. 1). Her functional and radiologic left leg length was 7 cm shorter than the right leg (Fig. 2) and the real length of the left femur and tibia was 4 cm shorter than the right side. This indicated that the flexion contracture emphasized the discrepancy. The preoperative neurovascular examination showed full motor and sensory function with palpable dorsalis pedis and posterior tibial pulses. Her feet were warm and well perfused clinically. All examinations were otherwise normal. The patient she

Fig. 1. Preoperative anteroposterior radiograph showing arthrodesed hip.

Fig. 2. Preoperative anteroposterior radiograph showing the total leg.
Obviously left leg is shorter than the right leg.

was evaluated by a vascular surgeon, a cardiologist, and an internist before the hip surgery. Noninvasive vascular studies revealed moderate bilateral lower extremity arterial occlusive disease.

A left side THA was performed using an anterolateral exposure to the hip. Uncemented press-fit cup fixation with additional screws fixation in the adequate manner and uncemented femoral component was used (MPF® Cup and SL Plus® Stems, Smith & Nephew AG, Rotkreuz, Switzerland). An extensive soft tissue release was necessary for the mobilization, including the proximal tenotomy of the adductors, the distal tenotomy of the gluteus maximums and the iliopsoas, the origin-release of the quadriceps and saltrius, and the periosteal release of the adductors from the femur. During the surgical procedures, the femoral and sciatic nerves were monitored by motor evoked potentials (MEPs) (Neuropack M-1, MEP9204, Nihon Kohden Corp., Tokyo, Japan)[11] with needle electrodes inserted in the rectus femoris and soleus muscles. The hip joint was reduced near the end of the surgery after the installation of all implants. The left hip developed a neutral position and mobility that had 60° of flexion, 0° of extension, 10° of abduction, 20° of adduction, 5° of external rotation, and 5° of internal rotation. Just after the reduction both the rectus femoris and soleus muscles showed no reaction on MEPs. The reaction of the contralateral hip was normal on MEPs. As nervous paralysis was suspected, the left hip was immediately dislocated again but no reaction was observed. Nothing more could be done except to add more soft-tissue-release and to reduce the joint. The leg was lengthened by 3 cm during the consecutive procedures as the length optimized soft tissue tension (Fig. 3). Just after awakening, the patient started to feel intolerable leg pain. Both of her dorsalis pedis and posterior tibial arteries were not palpable and her left leg was cool. An acute arterial occlusion was suspected, however this was not detected during the surgery because the middle of the thigh and the more distal leg were fully covered by thick surgical dressing. Blood flow in the femoral, popliteal, tibial, and dorsalis pedis arteries could not be estimated by Doppler ultrasound imaging of the left leg. The behavior on MEPs seemed ischemic not paralytic. An angiography was performed at 70 minutes postoperation, which revealed an occlusion of the left limb of the CFA (Fig. 4). The left CFA had been thrombosed and it accompanied the arterial stenosis of the vessel over 5 cm. A transcatheter thrombectomy was successfully performed through the opposite CFA. The angiographic findings, after the thrombectomy, indicated the deformity of CFA by longitudinal traction without vascular intimal tearing. Therefore, we suspected the leg lengthening and mobilization procedure had stretched the vessel and mechanical deformity had obstructed the vessel resulting in the formation of a thrombus. There was no apparent direct injury of the vessel caused by retractors or screw drill. After the thrombectomy, a slight blood flow was observed in flexed and adducted hip positions. The stenotic deformity of the artery was too severe to allow perfusion of the whole lower extremity and blood flow was completely interrupted when the hip was in the neutral position. The patient's

Fig. 3. Anteroposterior radiograph of left hip showing well-positioned uncemented implants.

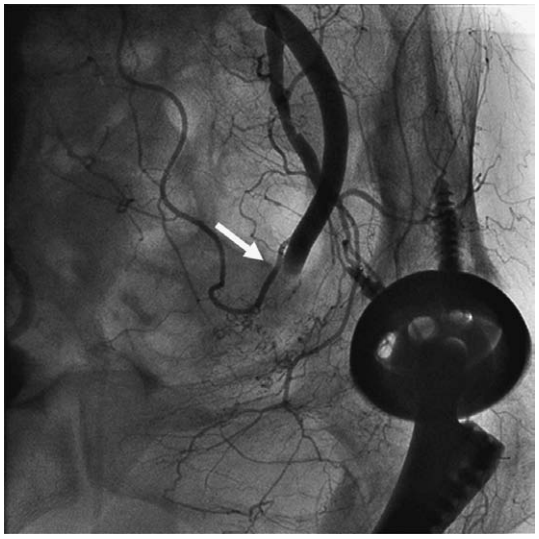

Fig. 4. Angiogram of left common femoral artery. The occlusion of the left common femoral artery during the arteriogram.

Fig. 5. Postoperative anteroposterior radiograph showing the total leg.
The leg length discrepancy was relativey well adjusted.

blood creatine kinase (CK-MB) value was 329 IU/L just after the surgery and was 368 IU/L after the transcatheter trombectomy. A stent placement was considered but the risk of stent fracture seemed high because of the stretch and stress of the vessel due to the hip mobilization. Therefore, an aortobifemoral bypass graft was performed at 210 minutes postoperation using a vascular prosthesis (Gelsoft® ERS Straight, Terumo Medical Corp., Tokyo, Japan) to improve the ischemia. Just after the vascular revascularization, the leg color was almost restored to normal (operative time was 1 hour 35 minutes). A significant

amount of bleeding was confirmed from the drainage tube in the hip joint because of release from soft tissues around the implants and from the anticoagulation treatment. Immediately after the aortobifemoral bypass graft, the hip was re-operated to arrest the hemorrhage using cautery, ligation, and absorbable topical hemostatlocal hemostatic (total operative time was 6 hours 7 minutes). Paralysis in the leg disappeared after the aortobifemoral bypass graft and the hemostatic procedure. The blood creatine kinase value was 601 IU/L at 10 hours after surgery.

At 4 months postoperation, the patient could walk with a single cane and had no apparent circulatory problems. She is fully satisfied with the result of the surgery (Fig. 5).

Discussion

Arterial injury after THA is relatively rare but is potentially limb-threatening[1]. Peripheral fibrosis secondary to previous surgery, infection, and advanced arthrosclerotic disease are all risk factors for vascular complications during total hip replacement[9, 12]. Reports in the literature indicate an average frequency of 0.2 to 0.3%[12-16] with prevalence varying from 0.08% for a single group of hip prostheses to 0.67%[15, 16] Complications can be delayed as in the case of pseudo-aneurysms and arteriovenous fistulas, or can result in immediate hemorrhage (sometimes with compressive hematomas) and/or ischemia from arterial injury, traumatic arterial tearing or mobilization of atheromatous plaque[17].

Duparc divided the lesional mechanism into three groups[18]. The dislocation or reduction maneuver of the joint can cause an indirect injury by vascular elongation or torsion. The persistent pressure from the tip of Hofmann type retractor placed on the anterior acetabulum wall can cause a direct extravascular type injury. The screws and/or drilling for the cup or perforation of the acetabular wall during acetabulum revision can cause a direct intravascular injury[18]. The diaceration of vessels during reaming and vascular complications secondary to an exothermic reaction during cement polymerization have also been reported[19]. Extraction of the acetabular anchoring cement plugs can also cause vascular wounds[20]. Shoenfeld *et al* reported 68 vascular complications associated with THA procedures. According to that report, the complications were caused by the incorporation of iliac vessels in cement in 30 cases, by the misplaced Hohmann retractor in 12 cases, by secondary to excessive traction on vessels in seven cases, by diverting from intrapelvic migration of an acetabular component in five cases, by excessive reaming in two cases, by osteotomy in one case, and by unknown origin in another 11 cases[21]. The presumed mechanism in the case reported here was excessive traction on the vessels. An unmovable hip (*e.g.*, arthrodesed hip) often has difficulties associated with reduction[1] and requires the stretching of vessels because of scarring around the vessels or abnormal shortening of the vessels.

Until this case we had replaced 14 hips after arthrodesis without any patients having severe perioperative complications. However, mobilization by means of THA for

arthrodesed hips is a challenging surgical procedure[22]. Arthrodesed hips have an increased risk of vascular complications because of the abnormal anatomy including contracture or shortening of vessels as well as angiectopia[1]. In recognition of the risk of vascular complications, precautions should be taken to minimize arterial injuries or thrombosis during THA surgery. Careful preoperative noninvasive assessment of vascular status is necessary, such as an ultrasound, duplex scan, or three-dimensional enhanced CT scan. Perforation must be avoided in the inner cortex of the acetabulum by acetabular reamers, drills, screws, or cement, especially in the anterior half of the acetabulum[23]. In addition, careful postoperative assessment is essential to detect signs and symptoms of limb ischemia at an early stage after THA. These precautions should be taken even in the case of ordinary THAs.

Fortunately, the affected limb was successfully salvaged in this case. According to Strathy and Fitzgecald, failure of THA is more common ($P < 0.05$) in patients who had a previous arthrodesis and in the patients who were fifty years old or less at the time of the arthroplasty[24]. Acute occlusion of the CFA is a serious event that can result in amputation or hip disarticulation. In this case, salvage of the limb was due largely to the early diagnosis of ischemia.

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